Anoxic hepatic and intestinal injury from carbon monoxide poisoning

Severe carbon monoxide poisoning results in widespread anoxic damage to many organs as a result of competition with oxygen for binding sites on the haemoglobin molecule and the consequent displacement of oxygen.[1 2] The anoxia principally affects the central nervous system and myocardium, although there has been one report of abdominal pain and bloody diarrhoea possibly as a result of anoxic damage to the intestinal wall.[3] We have been unable to find a description of anoxic hepatic injury sufficient to cause hepatic necrosis. This, together with severe intestinal symptoms suggestive of bowel infarction, was the principal clinical manifestation in the following patient.

Case report

A 24 year old woman had complained of a throbbing headache for three months before admission whenever she was in the kitchen. A gas water heater was subsequently shown to have a blocked flue. On the night of admission she had been working in the kitchen for one and a half hours when she developed a headache and felt faint. She could remember continuing to work there for a time but then became unconscious and was found two hours later. The total exposure time to carbon monoxide was three and a half hours.

When admitted to the local hospital 15 minutes later she was in deep coma, with a temperature of 33°C (rectal) and blood pressure 90/60 mm Hg. There was no jaundice, hepatomegaly, or focal neurological sings. Her skin was pink and the diagnosis of carbon monoxide poisoning was confirmed by a carboxyhaemoglobin saturation of 34% in the blood. Results of other investigations 12 hours after exposure were: haemoglobin concentration 17·7 g/dl, leucocyte count 33×10^9/l, platelet count 101×10^9/l, concentration of fibrin degradation products raised, prothrombin time 47 seconds (control 12 seconds), and blood urea concentration 9·1 mmol/l (56 mg/100 ml). No drugs were found in the serum and there was no history of excess alcohol ingestion. She was treated with 100% oxygen and fresh frozen plasma because of the prolonged prothrombin time. Twelve hours later she regained consciousness but then started to complain of abdominal pain, which was accompanied by loose stools containing blood. Forty eight hours after admission the pain had worsened and bowel infarction was suspected. At laparotomy the bowel was seen to be blue but viable. She failed to regain consciousness and it was found that the serum aspartate transaminase activity had been 1137 IU/l and the prothrombin time 55 seconds in a sample taken six hours before operation.

Next day, some 64 hours after admission, she was transferred to King's College Hospital, where she was found to be jaundiced with signs of grade 3 hepatic encephalopathy. Investigations showed that hepatic and renal function had deteriorated further—serum bilirubin concentration 60 μmol/l (3·5 mg/100 ml), aspartate transaminase activity 5000 IU/l, prothrombin time 45 seconds, urea concentration 18·0 mmol/l (108 mg/100 ml), creatinine concentration 510 μmol/l (5·8 mg/100 ml). Tests for hepatitis B surface antigen, IgM anti-HBc, and IgM antihepatitis A virus gave negative results. A renogram made with diethylenetriamine penta-acetic acid showed changes of acute tubular necrosis and she required haemodialysis for five days. By the seventh day after the incident her conscious level had returned to normal and the diarrhoea and abdominal tenderness had resolved. By the 11th day the serum aspartate transaminase activity and prothrombin time were normal. A biopsy specimen of liver obtained on the 15th day showed evidence of confluent cell loss with collapse of reticulum and accumulation of pigment macrophages. There was evidence of regeneration with prominent microvesicular fatty infiltration of the surviving parenchyma.

Comment

Usually a carboxyhaemoglobin concentration of 60% or higher is needed for coma to develop, and at a saturation of 34%, as in this patient, only confusion is present.[2] Once the patient is breathing uncontaminated air, however, the dissociation of carboxyhaemoglobin is initially rapid.[2] The carboxyhaemoglobin concentration in this patient was measured one hour after she had been removed from the contaminated room. Hence the peak concentration of carboxyhaemoglobin was probably higher and more than enough to account for her initial coma. Liver and bowel are sensitive to hypoxia.[4] In this case the hypoxia resulting from the carbon monoxide was sufficient to initiate the events that finally resulted in severe hepatic and bowel damage.

Surgery and anaesthesia reduce hepatic perfusion[4] and exacerbate diffuse parenchymal liver disease. This has been well documented after infectious hepatitis and in patients with cirrhosis who are operated on.[5] Without these added insults the hepatic necrosis may well have remained clinically latent in this patient, even though before laparotomy there was already evidence of severe hepatic necrosis (prothrombin time 55 seconds, serum transaminase activity 1137 IU/l). After laparotomy the clinical picture was of fulminant hepatic failure, although recovery was rapid. Hepatic damage has not been reported in carbon monoxide poisoning, but clinically latent forms may be more common than has been recognised hitherto and the possibility needs to be borne in mind during the management of these patients.

We thank Dr P B Portmann for the histological report on the liver biopsy specimen.

1 Jackson DL, Menzes MD. Accidental carbon monoxide poisoning. *JAMA* 1980;**243**:772-4.
2 Winter PM, Miller JN. Carbon monoxide poisoning. *JAMA* 1976;**236**:1502-4.
3 Hopkinson JM, Pearce PJ, Oliver JS. Carbon monoxide poisoning mimicking gastroenteritis. *Br Med J* 1980;**281**:214-5.
4 Powell-Jackson PR, Greenway B, Williams R. Adverse effects of exploratory laparotomy in patients with unsuspected liver disease. *Br J Surg* 1982;**69**:449-53.
5 Harville DD, Summerskill WH. Surgery in acute hepatitis. *JAMA* 1963;**184**:257-61.

(*Accepted 8 August 1984*)

Liver Unit, King's College Hospital and School of Medicine and Dentistry, London SE5
ALASTAIR WATSON, MA, MRCP, medical registrar
ROGER WILLIAMS, MD, FRCP, director of liver unit

Correspondence to: Dr Roger Williams.

Failure to thrive owing to inappropriate diet free of gluten and cows' milk

The hazards of nutritionally inadequate exclusion diets for infants have been outlined.[1] We describe an infant given such a diet on professional advice, whose consequent failure to thrive necessitated admission to hospital.

Case report

A 7½ month old boy presented with pallor and weight loss. He had been born at 39 weeks' gestation after a normal pregnancy and delivery, weighing 4100 g (above the 90th centile). He had been breast fed until 4 months old, when cows' milk and solids were added. His mother had sought advice because of his persistent crying during and after feeds, which was associated with some regurgitation but no diarrhoea. He also had facial eczema. She had been advised by her health visitor to replace cows' milk with a formula based on soybean and to withdraw food containing gluten. During the next six weeks he lost over 600 g in weight.

On examination he was thin and alert with wasting of the arms, legs, and buttocks. He had an eczematous rash on his left cheek and the napkin area. He weighed 6·3 kg (below the 3rd centile). His mucous membranes were pink. Investigations showed haemoglobin concentration 11·5 g/dl with a normal film and red cell variables; white cell count $14·7 \times 10^9$/l (37% neutrophils, 57% lymphocytes, 3% monocytes, 3% eosinophils); erythrocyte sedimentation rate 10 mm in the first hour; plasma concentrations of sodium 138 mmol(mEq)/l, potassium 4·7 mmol(mEq)/l, bicarbonate 18 mmol(mEq)/l, urea 7 mmol/l (42 mg/100 ml), creatinine 57 mmol/l (645 mg/100 ml), total protein 71 g/l, and albumin 49 g/l; and normal serum concentrations of IgG, IgA, and IgM. His urine was sterile, and no pathogens, ova, or cysts were isolated from his stools.

He was given a normal diet supplemented by breast feeding. Dietary assessment showed that his previous diet had been adequate in protein, iron, calcium, and vitamin C but had yielded a daily energy intake of only 0·4 MJ (97 kcal)/kg calculated on actual weight (equivalent to 0·3 MJ (72 kcal)/kg calculated on a weight corresponding to the 50th centile) owing to an inadequate intake of breast milk and soybean formula (maximum 227 ml daily). In addition, his solids had been restricted because his mother was unsure which foods contained gluten. In hospital his diet yielded a daily energy intake of 0·58 MJ (137 kcal)/kg calculated on actual weight (0·45 MJ (106 kcal)/kg for a weight corresponding to the 50th centile). He gained 600 g in weight in 12 days and was discharged. At 11 months old he had regained a position on the 50th centile for weight (10·0 kg).

Comment

This infant's dramatic increase in weight with a normal diet indicated that the inadequate intake of energy that he derived from